Case Report

Left Ventricular Assist Device Decommissioning: Challenging the Standard of Care

Grayson C. Eubanks, Christopher V. Chien, Elizabeth M. Volz, Mirnela Byku*

Department of Medicine, University of North Carolina, Chapel Hill, NC USA

*Corresponding author: Mirnela Byku. Department of Medicine, University of North Carolina, 6015 Burnett-Womack Building, CB 7075, Chapel Hill, NC 27599 USA.


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Abstract

A 48-year-old man with recovered left ventricular ejection fraction (LVEF) on HeartMate II Left Ventricular Assist Device (LVAD) support was admitted for driveline fracture. Hemodynamic evaluation with transthoracic echocardiogram and right heart catheterization at 6000 Revolutions Per Minute (RPM) was reassuring. After a failed attempt at percutaneous decommissioning, the patient underwent surgical ligation of the outflow graft. Two months after LVAD decommissioning, he was readmitted with an ejection fraction of 20% and cardiogenic shock. Our case challenges the concept of “no net flow” at 6000 RPM and questions if this is the correct speed to assess for myocardial recovery.

Keywords: Cardiac Assist Devices, Hemodynamics, Occluder, Systolic Heart Failure


Introduction

The number of LVAD implants continues to increase as advanced heart failure becomes more prevalent. Despite good medical therapy, true myocardial recovery is rare. Accurate assessment of underlying myocardial function with echocardiography and right heart catheterization is essential prior to LVAD decommissioning. Current data suggests that this assessment should be done at 6000 RPM.

Case Presentation

A 48-year-old man with non-ischemic cardiomyopathy (NICM) status post HeartMate II LVAD in 2014 presented to the hospital with dizziness and fatigue in the setting of low flow alarms due to a driveline fracture. He did not have evidence of volume overload on physical exam. Mean arterial blood pressure was 70 mm Hg. He was admitted to the hospital for evaluation of LVAD pump exchange versus LVAD decommissioning.

His medical therapy on LVAD support included metoprolol succinate 200 mg daily and lisinopril 10 mg daily. Echocardiograms for the past 3 years consistently showed a LVEF ≥50% and Left Ventricular End Diastolic Diameter (LVEDD) ranging from 4.0-4.5 cm. He had New York Heart Association (NYHA) Class I symptoms prior to his admission. He was not a candidate for cardiac transplant previously due to active nicotine and cannabis use.

To understand his potential for LVAD decommissioning, the patient underwent a speed study with transthoracic echocardiogram and right heart catheterization. His LVAD speed was gradually...
decreased from 9000 RPM to 6000 RPM. At 9000 RPM, his LVEF was 50-55% and LVEDD was 4.5 cm. Indirect Fick cardiac index (CI) was 2.79 L/min/m² and pulmonary capillary wedge pressure (PCWP) was 9 mm Hg. At 6000 RPM, his LVEF remained 50-55% with LVEDD at 4.5 cm. Fick CI was 2.03 L/min/m² and PCWP was 12 mm Hg. With a pulmonary artery catheter in place, the speed study was repeated 3 days later in the cardiac intensive care unit after spinning at 6000 RPM for 15 minutes. Results were also obtained after a 6-minute walk test. LVEF remained at 50%, LVEDD <4.5 cm, Fick CI 2.2 L/min/m², and PCWP <10 mm Hg. Following multidisciplinary conversation, the decision was made to decommission the LVAD and stabilize him with medical therapy while his modifiable contraindications for transplant were addressed.

Through a 7 French sheath in the right femoral artery, selective angiography of the outflow cannula was obtained using digital subtracted angiography by hand-injection of Omnipaque through a 6 French pigtail catheter. Based on the measurements from angiography, an 18 mm Amplatzer Atrial Septal Defect (ASD) Occluder was deployed under fluoroscopic visualization (Figure 1). Following deployment of the occluder device his LVAD was turned off. Blood pressure dropped to 70/40 and pulmonary artery systolic and diastolic blood pressures each increased by 10 mm Hg. Echocardiogram continued to demonstrate a normal LVEF. The occluder device was recaptured and the LVAD was turned back on at 6000 RPM with immediate improvement in blood pressure. Notably, with the speed at 6000 RPM, an LV angiogram showed mechanical unloading of his left ventricle, as evidenced by contrast exiting through the outflow graft, as opposed to no net flow (Figure 2, Supplemental Video 1). In light of these findings, percutaneous LVAD decommissioning was aborted. The patient returned to the cardiac intensive care unit with LVAD speed at 9000 RPM.

Figure 1: Angiography and deployment of 18 mm Amplatzer ASD occlude. (A) Angiography of the LVAD outflow cannula using digital subtracted angiography by hand-injection of Omnipaque. (B) Deployment of 18 mm Amplatzer ASD occluder in LVAD outflow cannula
Figure 2: Left ventricular angiogram at 6000 RPM. LV angiogram showing contrast in the LV (A) exiting through the outflow graft (B) with LVAD speed at 6000 RPM.

Supplemental Video 1
LVAD Unloading at 6000 RPM
LV angiogram showing contrast exiting through the outflow graft with LVAD speed at 6000 RPM

The following day, his LVAD abruptly stopped due to the driveline fracture and was unable to be re-started. He became acutely hypotensive with dyspnea and rising pulmonary artery pressures secondary to severe retrograde flow through the outflow graft with the LVAD no longer functioning. He was started on dopamine, intubated, and taken to the catheterization lab where the outflow graft was successfully occluded with a 25 mm PTS® sizing balloon (Figure 3) to eliminate the detrimental hemodynamic impact of retrograde flow. He continued to require dopamine to maintain adequate cardiac output and blood pressure.

Figure 3: Placement of PTS® Sizing Balloon in outflow cannula. Balloon occlusion of LVAD outflow cannula to halt retrograde flow.
Following multidisciplinary conversation, the decision was made to decommission the LVAD and stabilize him with medical therapy while his modifiable contraindications for transplant were addressed. Post-operatively he required support with dopamine and epinephrine, which were gradually weaned off over the course of two weeks. His repeat echocardiogram demonstrated an LVEF of 50% and a moderately dilated right ventricle with mildly reduced function. On the day inotropes were weaned off, blood pressure was 86/55, Fick CI 2.8 L/min/m2, pulmonary artery pressure 38/19 mm Hg, and central venous pressure 4 mm Hg. He was eventually discharged on metoprolol succinate 50 mg daily but could not tolerate other medical therapy. He was not on a diuretic.

At follow up several weeks later, hypotension continued to limit any additions to his medical therapy. One month later he was admitted to the hospital with cardiogenic shock. Echocardiogram showed an LVEF down to 20%. After stabilization with multiple inotropes, he eventually underwent successful HeartMate II explant and Heart Mate 3 implant.

Discussion

Consideration of LVAD discontinuation with explant or decommissioning begins with assessment of changes in ventricular structure and function. Despite appropriate medical therapy and mechanical unloading, no more than 10% of patients will respond to have an LVEF ≥40% and LVEDD ≤6.0 cm [1-3]. Even with improvements in LV size and EF, true myocardial recovery to allow for LVAD discontinuation is rare and occurs in only 1-2% of patients [2-4]. Available data suggests that patients with the highest chance of recovery and LVAD withdrawal include those that are young (<50 years old), have NICM, have been diagnosed within 2 years, and are not severely dilated (<6.5 cm) [3]. A recent study examining 40 patients with NICM and HeartMate II LVAD had success in identifying patients who would tolerate LVAD withdrawal based on echocardiographic and right heart catheterization findings [4]. With LVAD speed at 6000 RPM (no net flow) for 15 minutes, echocardiographic criteria for explant or decommission included a left ventricular end diastolic diameter (LVEDD) <60 mm and LVEF >45%. Right heart catheterization criteria included a PCWP <15 mm Hg and a resting CI >2.4 L/min/m2. Of 18 patients who met criteria for withdrawal within 18 months of LVAD implant, 16 remained free from mechanical circulatory support or heart transplant at 1 year. Similar criteria for LVAD withdrawal were used to determine recovery in a study in 2011 [5].

Despite meeting these criteria for LVAD decommissioning, aside from a mildly reduced Fick CI at 2.2, our patient acutely decompensated during the attempt at percutaneous occlusion and again 2 months after surgical ligation of the outflow graft. While the initial decompensation was likely secondary to incomplete occlusion of his outflow graft with the occluder device, his presentation 2 months later with cardiogenic shock revealed how dependent he was on the LVAD, with 6000 RPM likely giving him enough forward flow to prevent decompensation.

In conclusion, proper patient selection for HeartMate II explant or decommission remains difficult due to the rarity of the situation. Though several echocardiographic and right heart catheterization criteria at 6000 RPM have shown promising results regarding survival free from LVAD or transplant, our case questions these criteria and the idea of no net flow at this speed. Further studies are required to assess native myocardial function prior to HeartMate II withdrawal.

References